

PREPARED REMARKS
PUBLIC HEARING-US. HOUSE COMM. ON COMMERCE-SUBCOMMITTEES
ON HEALTH and ENVIRONMENT and on OVERSIGHT and INVESTIGATIONS
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ISSUE: Scientific Basis for EPA's Proposed Revisions to the National Ambient Air Standards for Ozone (O₃) and Particulate Matter (PM)

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AFFILIATION: New York University Medical Center

RELEVANT PERSONAL BACKGROUND

1. Academic Peer-Reviewed Research Incorporated into O₃ and PM Criteria Documents, which has been supported by the National Institute of Environmental Health Science, the Electric Power Research Institute, the Environmental Protection Agency, and the Health Effects Institute. This includes:
 - a) Respiratory tract deposition and clearance of airborne particles
 - b) Controlled human and animal inhalation studies of physiological responses to acidic particles
 - c) Field studies of population responses to air pollution exposures
 - d) Development and evaluation of air sampling and monitoring techniques
2. Academic Air Pollution Research Study Advisement
 - a) Member and Chair of External Advisory Comm., Harvard 6Cities Study (1978-1987)
 - b) Member of External Advisory Comm., Harvard - Health Canada - Multi-city Air Pollution Health Effects Study (1987-1991)
 - c) Chair of External Advisory Comm., USC - CA Air Resources Board Study of Effects of Air Pollution on Children (1992-present)
 - d) Chair of External Advisory Comm., Yale Univ.-Pierce Foundation Study of Health Effects of Kerosene Space Heater Effluents (1993-present)
3. Federal Agency Service on Committees Focussing on Inhalation Hazards
 - a) Chair, Clean Air Scientific Advisory Committee (CASAC) (1983-1987)
 - b) Member, CASAC Subcommittees on O₃ (1988-1997) and PM (1993-1997)
 - c) Chair, Physical Effects Review Subcommittee of Clean Air Act Advisory Council (1994-1997)

- d) **Chair, EPA Science Advisory Board (SAB) Review Committee for Risk Assessment for Environmental Tobacco Smoke (1991-1993)**
 - e) **Chair, SAB Review Committee for Risk Assessment for Dioxin and Related Compounds (1994-1997)**
 - f) **Chairman, Indoor Air and Total Human Exposure Advisory Committee, U.S. Environmental Protection Agency (EPA), 1987-1993**
 - g) **Co-Chair, 4th Task Force for Research Planning in Environmental Health Sciences, National Institute of Environmental Health Sciences (1992)**
 - h) **Chair, Board of Scientific Counselors, National Institute for Occupational Safety and Health (1990-1992)**
4. **International Service on Air Pollution Issues**
- a) **Chair of Working Group on Acute Health Consequences of Winter-type and Summer-type Smog Episodes. World Health Organization-European Region (1990-1991)**
 - b) **Member of Working Groups on Air Quality Guidelines. World Health Organization-European Region (1985-1987 and 1994-1997)**
5. **National Academy of Science Committees**
- a) **Member, Committee on Measurement and Control of Respirable Dust in Mines, National Materials Advisory Board, National Research Council, 1978-1979**
 - b) **Member, Committee on Toxicity Data Elements, Board of Toxicology and Environmental Health Hazards, National Research Council, 1980-1983**
 - c)' **Member, Committee on Methods for the In Vivo Toxicity Testing of Complex Mixtures from the Environment, Board on Toxicology and Environmental Health Hazards, National Research Council, 1985-1987**
 - d) **Member, Committee on Research and Peer Review in EPA, Commission on Geosciences, Environment, and Resources, National Research Council, 1994-1997**

OUTLINE OF REMARKS THAT FOLLOW

STATUTORY BASIS AND CASAC REVIEWS OF 03 AND PM DOCUMENTS

SCIENCE BEHIND EPA'S PROPOSED NAAQS REVISIONS

**General Issues
Particulate Matter
ozone**

FUTURE RESEARCH NEEDS

**Particulate Matter
Ozone**

MY RECOMMENDATIONS TO CONGRESS

STATUTORY BASIS AND CASAC REVIEWS OF O₃ AND PM DOCUMENTS

1. **Statutory requirement mandates the EPA to conduct periodic (nominally every 5 yrs) reviews of adequacy of National Ambient Air Quality Standards (NAAQS).**
2. **There is growing scientific peer-reviewed evidence for adverse human health effects at ambient concentrations below the existing NAAQS for PM (revised in 1987) and O₃ (last revised in 1979).**
3. **Extraordinarily thorough reviews of evidence were conducted by EPA, CASAC, and the public sector. They provide an open record for the EPA Administrator and Congress.**
4. **The following were strong CASAC consensus conclusions:**
 - a) **There is a need for more targeted indices of relevant exposure, e.g., 8-hr avg. O₃, and 2.5 µm cut-size for PM (PM_{2.5}).**
 - b) **There is a need for more robust criteria for daily NAAQS exceedances, i.e., multiple times rather than single.**
 - c) **Adverse health effects are occurring in U.S. communities currently in compliance with existing NAAQS.**
 - d) **Adverse effects are evident for sensitive subpopulations and may not as significantly affect most people (very large numbers of affected people, but low % of total population).**
 - e) **There are no identifiable threshold exposures for associations between PM and O₃ concentrations and adverse health effects.**
 - f) **PM_{2.5} and O₃ are largely formed in the atmosphere from gaseous precursors, are relatively uniformly distributed over large regions [hydrocarbons and nitrogen oxides (NO_x) react to form O₃, and organic components of PM_{2.5}; NO_x, SO₂ and photochemical oxidants react to form inorganic components of PM_{2.5} (sulfates and nitrates)].**
 - g) **Control strategies for PM_{2.5} and O₃ need to be implemented together, and on broad geographic scales.**
 - h) **Existing statutes and evidence presents difficult policy dilemmas to EPA Administrator and Congress because reducing PM and O₃ concentrations can reduce, but not eliminate, excess mortality and morbidity.**
 - i) **The ranges of the O₃, PM₁₀, and PM_{2.5} concentrations proposed in the final draft Staff Paper were appropriate choices for consideration by the EPA Administrator based upon an exhaustive review and evaluation of the peer-reviewed scientific data base.**
5. **Further Conclusions concerning the health effects of PM were drawn by CASAC Panel members with relevant experience in environmental epidemiology in a supplemental letter of 3/20/96 from Lippmann (New York Univ.), Shy (Univ. of N. Carolina @ Chapel Hill), Speizer (Harvard Univ.), and Stolwijk (Yale Univ.). The following are excerpts from this letter. The full text of the letter is published as Appendix H of PM Staff Paper.**

“In our judgment, the studies reviewed in the criteria document, specifically those considered in Chapter 12 (Epidemiological Studies), are persuasive in demonstrating a causal relationship between particulate air pollution, as measured by different methods in the various studies, and excess mortality and morbidity.

The reasons for concluding that particulate air pollution is causally related to excess mortality and morbidity are summarized here:

- **A large number (20) of epidemiological time-series studies have consistently found a statistically significant association between daily variation in particulates and total mortality in cities of the U.S., Canada, Latin America, the U.K., and continental Europe. These findings argue against the associations being attributable to statistical sampling variation, i.e., the role of chance.**
- **The results of these time-series studies cannot be attributed to the vagaries of statistical modeling, nor to confounding by season or weather.**
- **The results of the time-series studies cannot be attributed to other criteria air pollutants....Across the range of the 20 studies mentioned above, particulate air pollution is the only pollutant that is consistently associated with excess daily mortality, and the estimate of its effect is relatively stable when adjusted for the presence of co-pollutants....No monitored air pollutant, other than particulate matter, can account for the consistently observed excess mortality in these studies. Excess morbidity from cardiopulmonary diseases has also been observed in a considerable number of studies, and the morbidity relationship with ambient particulate concentrations is stronger overall and more consistent than for any other air pollutant.**
- **There is considerable coherence between the observed mortality and morbidity effects of particulate air pollution. Not only is excess mortality from cardiovascular and respiratory diseases observed, but on days of higher particulates excess hospitalizations for cardiovascular and respiratory diseases are reported....On days of high particulates, there is an increased proportion of deaths from chronic obstructive pulmonary disease, pneumonia, heart disease and deaths among the elderly than on days of low particulates. These findings are supportive of a causal role for particulate air pollution, since they are health endpoints one would most anticipate from exposure by the inhalation route.**

Given the striking consistency of the above studies, their robustness to variations in statistical modeling, the coherence among different but closely related health endpoints, and the empirical elimination of any alternative explanation for the findings, we conclude that a causal interpretation for particulate air pollution exposure is reasonable and defensible. This conclusion is further supported by longitudinal cohort studies of populations in which a geographical gradient in particulate air pollution was associated with a corresponding gradient in total mortality, in cardiopulmonary mortality and in lung cancer. These studies carefully controlled for other individual risk factors for these health endpoints.

Although population exposure to air pollution cannot be perfectly estimated based on central monitoring, these inherent errors in exposure estimation are more likely to cause an underestimation of the adverse health effects associated with pollution exposure, particularly in longitudinal cohort studies where individual risk factors and exposures are directly related to health effects. Thus the consistent positive findings cannot be attributed to exposure measurement error. Furthermore, there is growing evidence that fine particles are more uniformly distributed over large geographic areas than are coarse

particles, that measurements at one site give a reasonable estimate of the fine particulate concentrations across a city, and that fine particles penetrate and have longer lifetimes indoors than coarse particles. This evidence supports using ambient measures of fine particulates at a central site as an acceptable estimate of the average exposure of people in the community. For these reasons, we judge that uncertainties arising from air monitoring and human exposure estimation do not negate the consistent excess mortality and morbidity associations discussed above.

We believe that the case has been made that fine particulates, as measured by PM_{2.5}, are the best surrogate currently available for the component of particulate air pollution that is associated with excess mortality and morbidity....We are not claiming that PM_{2.5} is the causal agent, but rather that PM_{2.5} is a better measure than any alternative metric, of the complex in the particulate mass that is causing excess mortality and morbidity....Excess mortality, hospital admissions for respiratory diseases and decreased lung function are more strongly and consistently associated with fine rather than with coarse mode particulates.

The Health Effects Institute (HEI) reanalysis does not contradict any of the above conclusions. The HEI analysis conclusively demonstrated that the positive findings from the original studies selected for reanalysis were replicable, were not an artifact of statistical modeling, and were not confounded by idiosyncrasies in the method to control for season or weather....The HEI investigators appropriately concluded that, because of the high intercorrelations between pollutants in Philadelphia, mortality effects could not be attributed solely to particulates. More importantly, in their further report on this phase of their study, they concluded that "insights into the effects of individual criteria pollutants can be best gained by assessing effects across locations having different pollutant mixes and not from regression modeling of data from single locations."

In our judgment, EPA has appropriately synthesized this evidence and drawn a responsible public health conclusion, namely, that particulate concentrations at current levels are causally associated with excess mortality and morbidity. Furthermore, we agree that fine particulates, as currently indexed by PM_{2.5}, are the most appropriate indicator for the component of the particulate air mass to which these adverse effects are attributed. We also agree that some adverse health effects may be related to the coarse particulate mode, and that therefore it is desirable to consider fine and coarse mode particulates as separate candidates for air quality standards."

SCIENCE BEHIND EPA'S PROPOSED NAAQS REVISIONS

Generic Issues

Most scientific studies that are relevant to the setting of NAAQS were not designed or performed with that specific application in mind. Some research conducted in EPA laboratories or performed elsewhere by EPA contractors or grantees did have such applications in mind, but such EPA-supported research has been far too limited in scope, nature and extent to provide a data base for standard setting, especially for PM where much of the critical information has come from epidemiological research. The fact is that, because of limited research resources, constantly shifting research priorities, and a long-term policy choice to have only a minimal in-house capability for epidemiologic research, the bulk of the health effects research most relevant to a PM NAAQS has been performed by academic investigators with resources provided by others such as, for example, the National Institutes of Health, the Health Effects Institute, the Electric Power Research Institute, Health-Canada, and the California Air Resources Board. One result of this welter of diverse sponsorship, and therefore of research goals, is a wealth of information that is, unfortunately, composed of bits and pieces of the overall puzzle. It requires

careful sifting to separate those elements of sufficient quality to inform the issues, as well as mature judgment to fit the pieces into an informative framework sturdy enough for summary judgments.

The incredibly careful sifting of the evidence performed by EPA's National Center for Environmental Assessment (NCEA), under the oversight and prodding of CASAC, ensures that essentially all of the relevant peer-reviewed science is examined in detail and appropriately summarized and interpreted in the final draft of the Criteria Document (CD). The corresponding public review sessions by CASAC of the Staff Paper (SP) drafts prepared by EPA's Office of Air Quality Planning and Standards (OAQPS) also ensures that the final draft of that document provides appropriate summary judgments on the scientific aspects of those items in the CD most relevant to the setting of the NAAQS. These items include the effects of concern, populations at special risk, optimal form and averaging times for the NAAQS, and the most likely residual effects associated with exposures to be expected across a possible range of concentration limits. This process thus provides the Administrator with the best possible basis for the difficult NAAQS decisions that are required to be made periodically under mandate of the CAA amendments of 1977.

There has never been a decision point where the Staff, the CASAC, or the Administrator has been satisfied with the available scientific data base, despite the ever increasing size and sophistication of the available data in successive review rounds. Our current knowledge always leads to new questions and concerns. More so than in the past, we are debating whether the measurable effects are sufficiently adverse to warrant public health protection rather than identifying whether measurable effects are occurring. However, the Administrator will still have, and will, I suspect, always have to make a judgment as to the margin of safety to apply in the absence of definitive knowledge.

Despite all of their thoroughly discussed and acknowledged limitations, the PM and O₃ literature reviews and analyses in the CDs and SPs are the best prepared and most comprehensive ever available to an Administrator as a basis for NAAQS decisions. In fact, the favorable contrast of these CDs and SPs with those from prior rounds of NAAQS is really remarkable.

Particulate Matter

Despite the consistent and coherent epidemiological evidence for very large impacts of ambient PM concentrations that do not exceed the current NAAQS, there are many who maintain that the promulgation of the proposed PM_{2.5} NAAQS would be premature and/or unwise as public policy. Their objections seem to fall into five main categories: 1) the relative risks, while generally significant statistically, are too low to be convincing, and there must be some unmeasured confounding factor(s) that, if known, would account for the associations; 2) no known biological mechanism(s) can account for the effects; 3) there may be an undetected threshold concentration and it is important to know the curvature, if any of the exposure-response relationship prior to establishing a NAAQS; 4) the costs and/or the societal disruptions of implementing the PM_{2.5} NAAQS are too great in relation to the benefits; and 5) that the CASAC did not endorse the PM Staff Paper and the PM_{2.5} concentration range in it. I will deal with each of these objections in turn.

Unidentified Confounders: The search for confounding factors that could account for the PM-effects associations has been intensive over the past five years. Factors examined and found not to be consistently influential include other criteria pollutants, temperature, humidity, and synoptic weather. It thus seems highly unlikely that confounding can explain the coherent PM-effects associations.

Unknown Mechanisms: There are other well known examples where associations have become generally credible in the absence of well-established mechanisms. The most relevant example is environmental tobacco smoke (ETS), which also has produced highly significant associations with respiratory morbidity in children and comparable evidence for excess lung cancer. In both cases, the dominant sources are known, i.e., indoor smoking for ETS and fuel combustion (both stationary and motor vehicles) for PM_{2.5}, and we know how to control them.

Threshold Concentrations: What is clear is that if a threshold does exist, it has to be well below the proposed PM_{2.5} NAAQS. Thus, this is clearly a moot point.

Cost vs. Benefits: The PM Staff Regulatory Impact Assessment, using analytical techniques thoroughly reviewed and endorsed by the federally established Clean Air Act Compliance Analysis Council, has estimated the annual costs of compliance in 2007 will be $\$6 \times 10^9$ while the benefits will be in the range of $\$58-119 \times 10^9$. The largest component of the benefits is for premature mortality avoided and is greatly dependent on the value selected ($\$4.8 \times 10^6$) for premature mortality. Even if a much smaller value was used for this outcome, the benefits would still substantially exceed the costs.

CASAC Endorsement: After endorsing the PM Staff Paper as an effective summary of the relevant scientific data for setting revised PM NAAQS, the members of the CASAC PM Panel were asked by the Chairman to state their personal preferences for PM_{2.5} concentration limits. Of the 21 members, only two did not believe that a PM_{2.5} NAAQS was needed. Three others felt that the daily PM_{2.5} limit should be no more stringent (on average) than the current daily PM₁₀ limit, and therefore recommended a value outside the range proposed in the Staff Paper. On the same basis, two members recommended annual PM_{2.5} concentration limits outside the Staff's range. My interpretation is that the Administrator received a general, if not unanimous, endorsement of the Staff's PM_{2.5} recommendations.

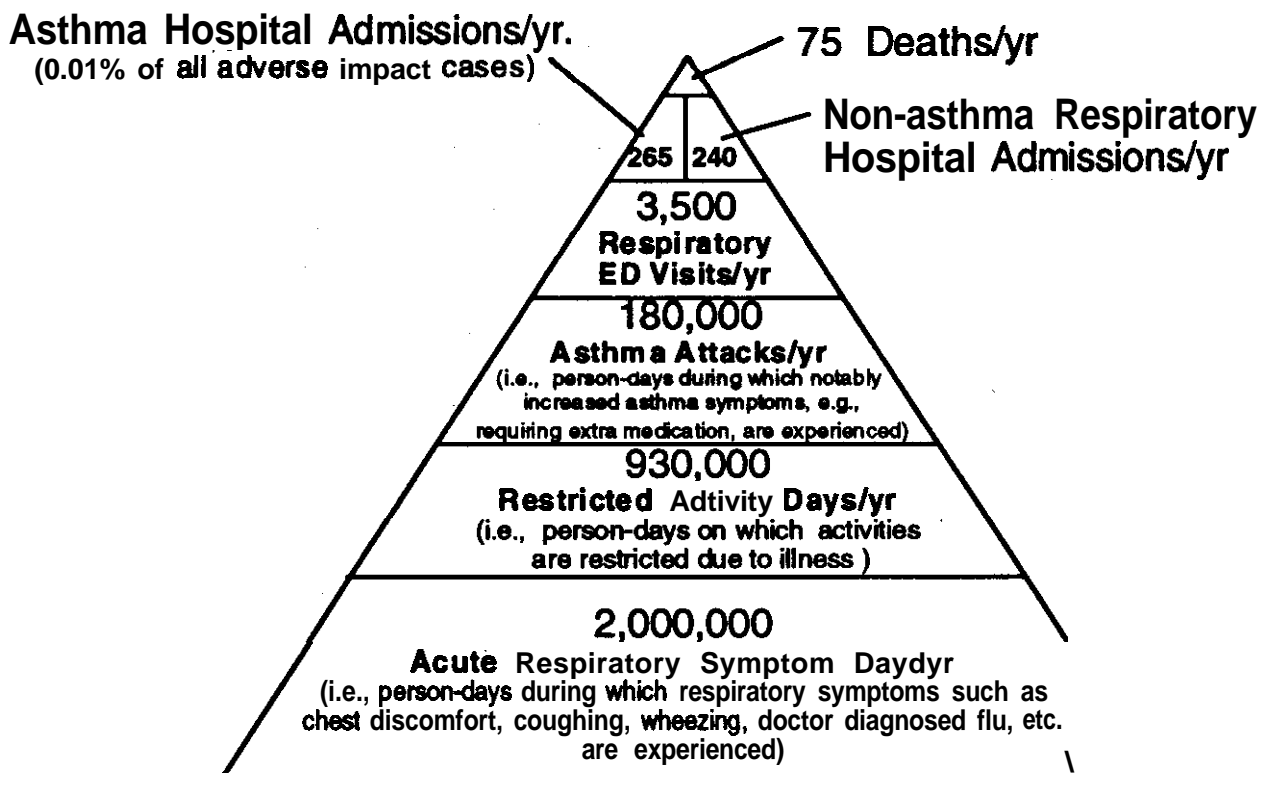
In summary, the science base for the revised PM NAAQS, while inevitably incomplete, is voluminous and highly informative in its totality. It has been carefully and thoroughly analyzed by EPA Staff and vigorously reviewed by CASAC. The PM Criteria Document and Staff Paper have CASAC endorsement in terms of their usage of the literature and data. The Administrator not only should complete the long and laborious process of revising the PM NAAQS based on these reviews and recommendations, but has the legally mandated requirement to do so. In my view, the proposed PM NAAQS is clearly not too strict. In terms of the selection of an improved index of relevant exposures and a modest degree of greater public health protection, it is a prudent judgement call by the Administrator. The proposed PM limits may not be strict enough to fully protect public health, but there remain significant knowledge gaps on both exposures and the nature and extent of the effects that make the need for a more restrictive NAAQS difficult to justify at this time. It is essential that adequate research resources be committed to filling these gaps before the next round of NAAQS revisions.

Ozone

The 03 Criteria Document (CD) thoroughly summarizes the myriad well-documented health effects that occur in both healthy people and asthmatics as a result of exposure to O₃ in ambient air, including pulmonary function deficits, lung inflammation, increased lung permeability and responses to allergic stimuli, altered lung clearance of inhaled particles and increased infectivity of disease agents, increased rates of usage of clinics, emergency rooms and hospital beds for respiratory diseases, and lost-time from work and school. It also discussed equivocal evidence for excess daily mortality on peak O₃ days. More recent positive findings in peer-reviewed papers on studies in London, England; Rotterdam and Amsterdam in the Netherlands; and Brisbane, Australia increase the likelihood that O₃ exposure does indeed cause excess mortality.

The O₃ Staff Paper evaluated data from a NYU study of excess daily hospital admissions for asthma in New York City (of which I was a co-author) as a key example of an adverse health effect of O₃ exposure. The tabular summary of this analysis in the Staff Paper indicated that the number of asthma admissions attributable to O₃ was a relatively small fraction of the total number of year-round asthma admissions. This is certainly true, but ozone is a summertime phenomenon, and the year-round denominator is therefore not the appropriate divisor. Asthma is a serious and growing problem to millions of people and the health-care community. There is no good evidence that O₃ causes new cases of asthma, but clear evidence that it exacerbates the condition in the numerous people who suffer from asthma.

Pyramid of New York City, NY Annual Adverse Ozone Impacts Avoided By The Implementation of The Proposed New Standard (vs. "As Is")



*Figure section sizes not drawn to scale.

What is not evident from the analyses in the Staff Paper is that the hospital admissions for asthma is not the only, or even the most serious of the adverse impact of O₃ on human health. Rather, it serves as the "lamppost" under which the evidence was most readily visible. My colleague at NYU, Dr. George D. Thurston, has prepared a visual aid, based on his research and research by others, to more fully illustrate the range and magnitude of the health effects attributable to O₃ in New York City in each year that could be avoided by implementation of the proposed revision of the O₃ NAAQS. It can be seen that the estimate of 265 hospital admissions for asthma is near the tip of the "iceberg", along with 240 other hospital admissions (for other pulmonary diseases), 75 cardiopulmonary deaths, and 3,500 emergency room visits. It also can be seen that the total impact extends to millions of excess symptoms and disease incidences.

FUTURE RESEARCH NEEDS

While the present research base is more than adequate to support the current EPA NAAQS proposals, more can be learned that can aid in the most efficacious implementation of the new standards during the next decade, as well as to provide a basis for still better focussed NAAQS in the next round.

Particulate Matter

I had the opportunity to chair an informal 1-1/2 day workshop devoted to research needs on the health effects of airborne PM in Park City, Utah on April 29 and 30, 1996. It was held in conjunction with the Second Colloquium on Particulate Air Pollution and Health at Park City, Utah, on May 1-3, 1996. The objective of the Workshop was to prepare a holistic assessment of knowledge gaps and research opportunities for presentation at the penultimate session of the Colloquium. The Workshop reviewed the research progress made since the first PM Colloquium (Irvine, CA - January 1994) and the findings of recent major reviews of the PM literature by WHO-EURO, U.K. Health Department, RIVM in the Netherlands, and the U.S. EPA. It then discussed: 1) the nature of ambient PM; 2) population segments at special risk; 3) the nature of the health effects of concern; 4) the sources of ambient air PM; and 5) the implications of ambient air PM health effects on occupational exposure limits and occupational cohorts. The Workshop concluded that:

- A primary focus for further research should be on accumulation mode aerosol with the objective of disentangling the roles of its chemical constituents, as well as their interactive effects with each other and with co-existing gaseous criteria pollutants.
- Research is also urgently needed on the health effects of both the coarse mode PM₁₀ and the ultrafine particles in the nuclei mode aerosol.
- There should be a continued focus on populations of special concern, i.e., Infants, the elderly, and people with pre-existing cardiopulmonary diseases.
- Further development and validation of animal models for human sensitive groups warrants high priority.
- Validated animal models are needed for target human populations in order to investigate: a) the roles of specific constituents of PM mixtures; b) the roles of exposure concentrations and durations on responses; c) some of the risk factors that predispose individuals to be responsive to PM exposures; d) physiological, biochemical, molecular and pathological correlates of mortality, tissue and organ damage, and chronic disease development.

The full, peer-reviewed, manuscript outlining these research recommendations in greater detail is in press in the scientific journal "Applied Occupational and Environmental Hygiene."

Ozone

In terms of research needs for O₃, the following are my personal recommendations based upon my own research experience and service on CASAC panels.

The most pressing need is for research on the cumulative effects of O₃ on lung development in children and on accelerated aging of lung structures that may shorten life-span in adults. We have a lot of data on transient functional effects of O₃ from controlled human exposure studies. -Such studies can provide information on chronic pollutant effects only to the

extent that prior exposures affect the transient response to single exposure challenges. Most of the limited data we have on the effects of chronic O₃ exposures on humans come from epidemiological studies. Epidemiological studies can establish chronic health effects of long-term O₃ exposure in relevant populations, and offer the possibility that the analyses can show the influence of other environmental factors on responses to O₃ exposure.

We also need more controlled exposure studies focussed on the mechanisms and patterns of response to inhaled O₃ and of the influence of other pollutants and stresses on these responses. Studies of the transient responses to acute exposures can establish the interspecies differences in response among animal species, and between them and humans similarly exposed. Animals are needed for studies of responses that require highly invasive procedures or serial sacrifice to gain information that cannot be obtained from studies on human volunteers. Finally, we should use long-term exposure protocols in animals to study cumulative responses and the pathogenesis of chronic disease in animals. Studies on animals can examine the presence and basis for variations in response that are related to age, sex, species, strain genetic markers, nutrition, the presence of other pollutants, etc.,

Research is also needed to establish the interrelationships between small transient functional decrements, which may not in themselves be adverse effects, and changes in symptoms, performance, reactivity, permeability, and counts of inflammatory cells. The latter may be closely associated with adversity in themselves, or in the accumulation or progression of chronic lung damage.

Chronic human exposures to ambient air appear to produce a functional adaptation that persists for at least a few months after the end of the O₃ season but dissipates by the following spring. Several population-based studies of lung function have indicated an accelerated aging of the lung associated with living in communities with persistently elevated ambient O₃. The plausibility of accelerated aging of the human lung from chronic O₃ exposure is greatly enhanced by the results of chronic animal exposure studies in rats and monkeys. There is little reason to expect humans to be less sensitive. Humans have a greater dosage delivered to the respiratory acinus than do rats for the same exposures. Also, the rat and monkey exposures were to confined animals with little opportunity for heavy exercise. Thus, humans who are active outdoors during the warmer months may have greater effective O₃ exposures than the test animals. Finally, humans are exposed to O₃ in ambient mixtures. The potentiation of the characteristic O₃ responses by other ambient air constituents seen in short-term exposure studies in humans and animals may also contribute toward the accumulation of chronic lung damage from long-term exposures to ambient air containing O₃.

In summary, the lack of a more definitive data base on the chronic effects of ambient O₃ exposures on humans is a serious failing that must be addressed with a long-term research program.

MY RECOMMENDATIONS TO CONGRESS

1. Recognize that EPA Administrator has made a prudent public health judgment in her PM and O₃ NAAQS selections.

The health benefits (cost avoidance) to be derived by implementation of the new PM_{2.5} NAAQS will far exceed the costs of control implementations. The benefit/cost ratio for implementing compliance with the revised O₃ NAAQS is not as great, but it should be recognized that reductions in O₃ formation will also reduce PM_{2.5} formation and ambient air concentrations and will also therefore contribute to the benefits associated with reductions in PM_{2.5} exposures.

For O₃, the current NAAQS of a 1-hr max of 120 ppb not be exceeded more than 4 times in 3 yrs is equivalent to an 8-hr max of 90 ppb based on the 3rd highest 8-hr value in a

year. Thus, the proposed 8-hr max of 80 ppb is only a modest O₃ NAAQS reduction. By contrast, the Air Quality Guideline for O₃ of the World Health Organization-European Region (WHO-EURO), adopted late in 1996 is an 8-hr maximum of 60 ppb. In my view, the 8 hr-80 ppb proposal is a prudent step in the right direction at this time and recognizes that any lower limit is probably not achievable without draconian controls. The major advance is the shift to an 8-hr averaging time, providing a much sounder basis for evaluating the public health risk from community exposures.

For PM₁₀, the 50 µg/m³ annual average would be retained without change, and the 24-hr PM₁₀ would be relaxed by applying it only to the 98th% value (8th highest in each year, averaged over 3 years) rather than to the 4th highest over 3 yrs. It is only by implementing the new PM_{2.5} NAAQS that the degree of public health protection would be substantially advanced by focusing control efforts on sources of fine particles. Such controls are not effectively addressed when only PM₁₀ is regulated.

2. Recognize that 1990 CAA-Title I implementation already underway (SO₂ and NO_x emission reductions) will reduce the numbers of communities in exceedance of the proposed PM₂₅ NAAQS relatively soon
3. Recognize that the new PM_{2.5} NAAQS cannot be implemented immediately, and prudent implementation schedules can be devised and implemented to minimize economic disruptions.
4. Recognize that the causal factors within PM_{2.5} for the consistent and coherent associations between PM_{2.5} in community air and excess daily and annual mortality, excess emergency room and hospital admissions for respiratory diseases, lost time from work and school, respiratory symptoms, and reduced lung functions are not yet fully established in terms of biological mechanisms. However, it has clearly been shown that they cannot be attributed to other hypothesized environmental factors such as other criteria air pollutants, aeroallergens, or meteorological variables. The situation is analogous to that for another commonly encountered respiratory irritant, i.e., environmental tobacco smoke, where the epidemiological evidence for adverse respiratory effects in children is overwhelming, and there is significant evidence for excess lung cancer in adults as well.
5. Recognize that more definitive laboratory and epidemiological research on causal factors is now becoming feasible as epidemiologic investigative techniques and animal models for susceptible segments of the population are being established and validated. With a reasonable and prudent level of additional research funding for EPA and NIEHS, identification of the biological mechanisms, the chemical and physical properties of the active components of PM, and the exposure-response relationships, can be more firmly established within the next five years. Such knowledge is essential for the design and implementation of cost-effective control strategies, and for the further revisions of the PM NAAQS that will be required early in the next century.
6. Recognize that while the costs of the research recommended above are substantial (on the order of \$50x10⁶ per year), they are quite small in relation to the control costs that can be more effectively targeted and reduced through the knowledge gained, and also small in comparison to the health benefits resulting from exposure reductions resulting from the implementation of the revised NAAQS.

SUMMARY OF ORAL REMARKS
PUBLIC HEARING-U.S. HOUSE COMM. ON COMMERCE-SUBCOMMITTEES
OVERSIGHT ON HEALTH AND ENVIRONMENT,
AND OVERSIGHT AND INVESTIGATIONS

April 10, 1997

ISSUE: Scientific Basis of the EPA's Proposed Revisions to the National Ambient Air Quality Standards (NAAQS) for Ozone (O₃) and Particulate Matter (PM)

SPEAKER Morton Lippmann, Ph.D., Professor of Environmental Medicine, New York University Medical Center

Major Points

1. Most scientific studies that are relevant to the setting of NAAQS were not designed or performed with that specific application in mind, and for PM where much of the critical information has come from epidemiological research, the bulk of it was performed with resources provided by others than EPA.

2. The wealth of information is composed of bits and pieces. It requires careful sifting to separate those elements of sufficient quality to inform the issues, as well as mature judgment to fit the pieces into an informative framework sturdy enough for summary judgments.

3. Very careful sifting of the evidence has been performed by EPA under the oversight and prodding of CASAC, ensuring that essentially all of the relevant peer-reviewed science has been examined in detail and appropriately summarized and interpreted in the Criteria Documents (CDs).

4. The Staff Papers (SPs) provide appropriate summary judgments on the scientific aspects of the issues most relevant to the setting of the standards.

5. This process provides the Administrator with the best possible basis for the difficult NAAQS decisions that are required to be made periodically under mandate of the CAA amendments of 1977.

6. There has never been a decision point where the Staff, the CASAC, or the Administrator has been satisfied with the available scientific data base, despite the ever increasing size and sophistication of the available data in successive review rounds. However, despite all of their thoroughly discussed and acknowledged limitations, the PM and O₃ literature reviews and analyses in the CDs and SPs are the best prepared and most comprehensive ever available.

7. Objections raised by others do not warrant any extended delay. The new PM2.5 **NAAQS** is essential to the development and implementation of source controls on fine particle precursors and the reduction of the adverse health effects resulting from current exposures.

a. **For** ozone, the chart on page **8** of my remarks was based on NYU research in hospital admissions in New York City and a variety of studies elsewhere by us and others. The study analyzed in detail in the EPA Staff Paper on asthma admissions to New York City hospitals is just one, relatively small component of the quite large overall human health impact of current ozone exposures, and only for one city.

9. There are important unresolved issues at the end of each review round that should set in motion a substantial research program. We will need to invest at least **50** million dollars a year over the next five years in **NAAQS** research. This investment will pay **for** itself many times over **in** terms of an enhanced ability to target air pollution source controls on the most important emissions, as **well** in terms of improvements **in** public health and savings in health care resources.